FETAL ASCITES AS A CAUSE OF DYSTOCIA IN LABOUR

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Fetal AsciteS, a relatively uncommon problem, is now being diagnosed more frequently following the introduction of routine ultrasound scanning during pregnancy[1-4]. As a cause of dystocia in labour, however the condition is very rare[5,6] and not many cases have been reported in literature[7-13], Three centuries ago Mauriceau (1681) described his difficulties in delivering a woman of an infant whose abdomen was distended by 5 quarts of fluid and Dorland (1919) described a no less horrifying story[14] Such cases are, however, rarely encountered now in the developed world. In the dept. of Obstetrics and Gynaecology- Jinnah Postgraduate medical centre, 2 cases causing dystocia seen over a 12 month period are described here.

CASE-1

The patient was a 30 year old para 1+1 with no living children. Her first pregnancy out of a first cousin marriage 2 years back had resulted in spontaneous vaginal delivery ofa fresh stillborn malformed infant at 32 weeks for which no cause could be ascertained. Her second pregnancy ended in a spontaneous abortion at 12 weeks again with no detectable cause.

During her current pregnancy, she was first seen in the of obstetrics and gynaecology, JPMC at 24 weeks gestation. An ultrasound examination done for date/size discrepancy showed a fetus corresponding to 25 weeks gestation having oedema of scalp and body wail alongwith massive ascites. The movements were sluggish and the placenta was low lying completely covering the os. Laboratory study revealed Hb 11.5 gms and no abnormality on urine analysis. Blood group was A + ye with no atypical antibodies; VDRL and KT were negative and blood urea and sugar levels were normal. Antibody titres to TORCH infections were insignificant. Vaginal termination of pregnancy at this stage was not possible because of the lowlying placenta. The patient refused any hospitalization and was advised to report for periodic antenatal examination, keeping in mind placental migration and subsequent vaginal delivery.

She was then lost to follow up and reported at term in labour for 6 hours. On admission she had slight pedal edema anda bloodpressure of 120/70mm Hg. The fundal height was large for dates and the fetus was presenting as head which was 3/5 palpable per abdomen. The fetal heart sounds were absent. Vaginally the os was 4 cms dilated with a 50% effaced cervix and head at -2 station.

Membranes were intact and the pelvis was adequate. Abdominal paracentesis of fetal ascites was attempted but it failed. Labour was allowed to proceed. Membranes ruptured spontaneously and she approached full dilatation 6 hours later. The head descended to +1 station and outlet forceps were applied for poor maternal effort. Despite the application and extraction being easy, the head was detached from the body. Thereafter all attempts at decompression of the fetal abdomen both per abdomen and per vaginum failed. Vaginal delivery was then abandoned and abdominal delivery was decided. An immediate lower segment Cesarean section was performed. 3 litres of ascitic fluid was drained from fetal abdomen and the body was delivered.

The baby was a 3.2 kg hydropic male, macerated and stillborn. Other than a massively distended abdomen and an enlarged liver, there were no apparent abnormalities. Autopsy was not done. The placenta was large and edematous, weighing 1.2 kg. His topathologi cal examina

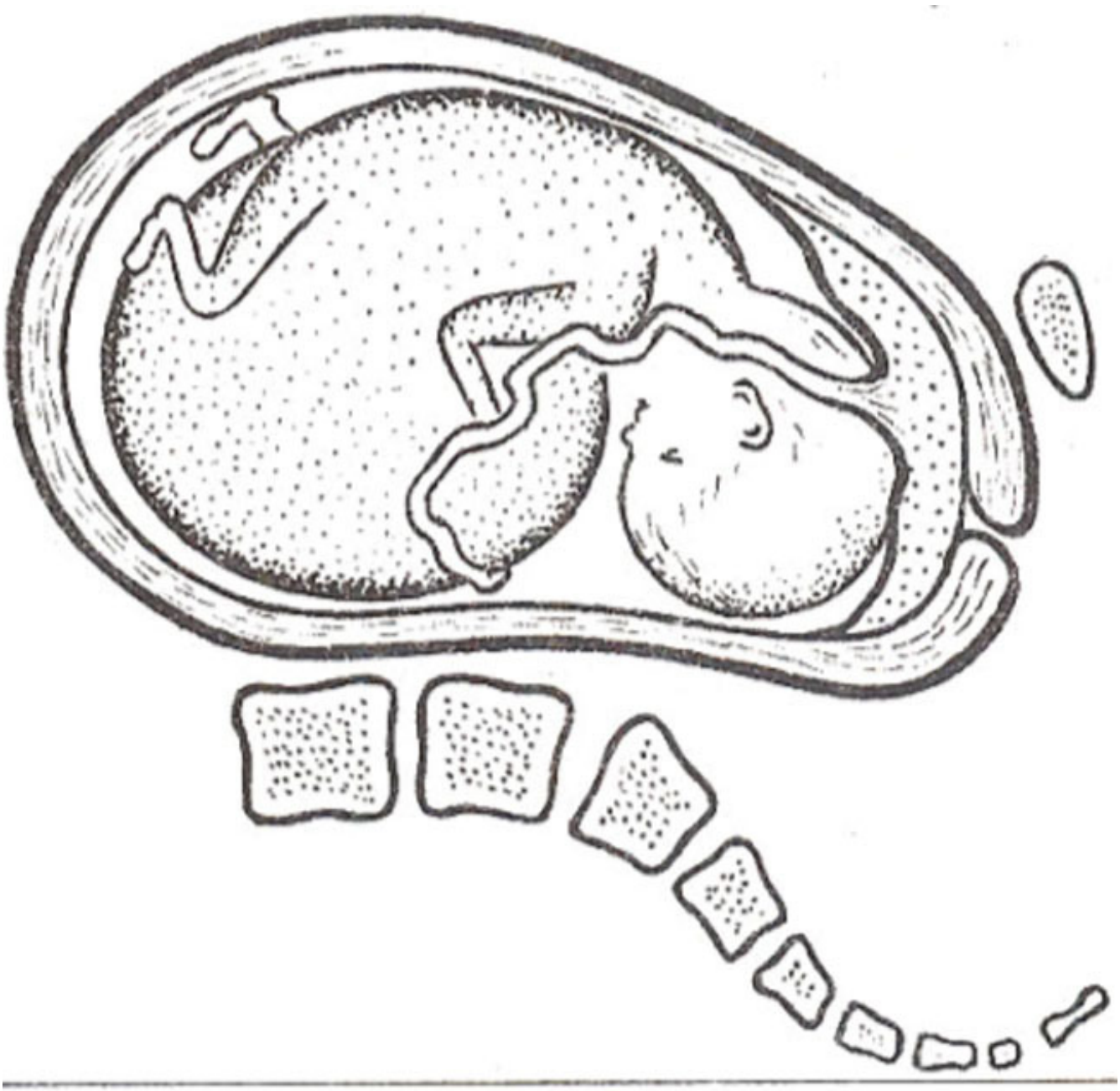

Figure 1. Placenta at 24 weeks gestation covering the os.

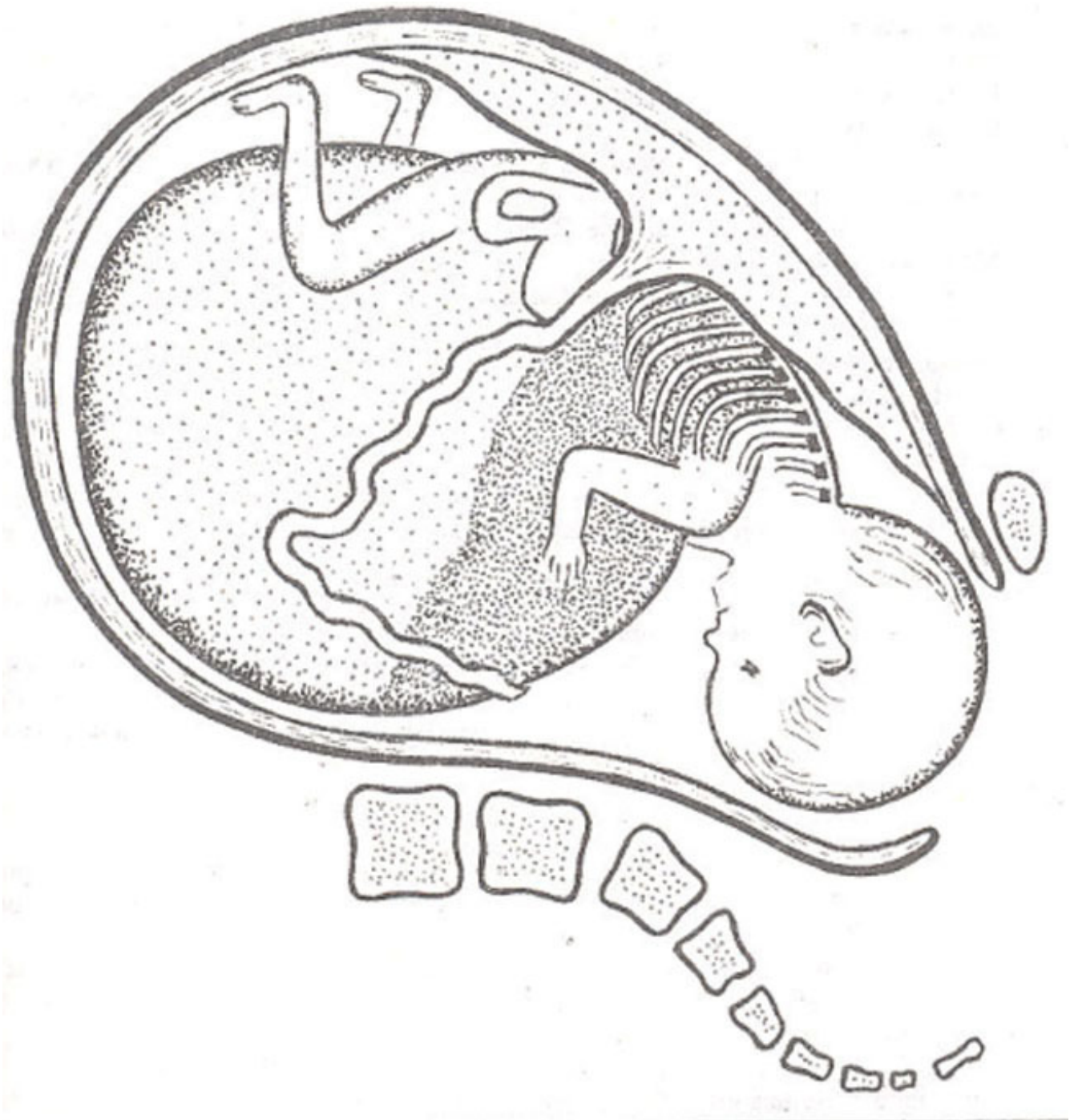

Figure 2. Fetal position in utero that prevented paracentesis of ascitic fluid.

Figure 1 Placenta at 24 weeks gestation covering citeostion did not reveal any significant change.

CASE-2

A 25 year old primigravida reported in labour at 39 weeks. gestation. The marriage was consanguineous, husband being her first cousin. She had no regular prenatal care, but had been to a practitioner a week prior to admission who had advised an ultrasound examina¬tion. The report showed fetus presenting by breech with marked ascites. There were no other associated fetal abnormalities. Her Hb was 10.00 gms and blood group was 0 +ve.

On admission she had been in labour for 3 hours. She was mildly anaemic; pedal edema was slight and blood pressure was normal (120/80mm Hg). Abdominal examination revealed uterus large for dates

with fetus presenting by breech and absent fetal hearts. Vaginally the os was 4 cms dilated with breech at -1 station. Membranes ruptured during examination draining meconium tinged liqour. The pelvis seemed adequate.
Paracentesis of fetal ascites through maternal ab¬domen yielded 250 cc fluid. The patient approached full dilatation 7 hours later. After the delivery of legs, dystocia was encountered. A wide bore needle was passed into the fetal abdomen vaginally and 1500 cc ascitic fluid was drained. Thereafter the delivery was accomplished un
eventfully.
The infant was a 3.8 kg female, fresh stillbirth. Except for abdominal distension, no other abnormality was detected. Autopsy was not done. The placenta apparently appeared normal.
The patient left against medical advice the next day.

DISCUSSION

Fetal Ascites is an infrequent and often a fatal phenomenon[1]. Itusuallypresents either as aprecurser or one of the findings in fetal hydrops[4,15,16]. The reported incidence varies between 1:2500 to 1:3748[15,17] births. However, only a few of these cases reach massive proportions and an insignificant number cause dystocia in labour.
The etiological factors may be immunologic as the result of isoimmunization[2,4,15,18] or non-immunologic often associated with fetal abnormalities[15,18]. Major categories include gastrointestinal[2,17,19], portohepatic[2,5,20], genitourinary[2,15,17]. and cardiovascular anomalies[2,3,15,18,21]. It has also been found in association with infections[2,15,17,22,23]. neoplasia[8,9,21], chromosomal[15]. haematological disorders[15,16]. lymphangectasias[2] multiple gestations[15,17,18] and placental chorangiomas[17] or the etiology may be indeterminate[2,15,17]. After an adequate diagnostic workup the cause can be ascertained in 84% cases[15], genitourinary and gastrointestinal anomalies together accounting for over 60% of the reported cases[2].
The pathophysiology of intra-pentoneal fluid accumulation is obscure, but the postulated mechanisms include chronic intrauterine anaemia, intrauterine congestive heart failure with obstructed venous return, hypoproteinemia or a combination of these[16,24,25].
Certain conditions have been affirmed as good indices of suspicion of fetal ascites i.e., polyhydramnios, maternal anemia, pre-eclampsia and diabetes mellitus[14,18,26]. The condition appears commonly in young primigravidae and a proportion of women present with antepartum haemorrhage, maipresentations and preterm labour[7,16]. Third stage complications such as retained placenta or primary postpartum haemorrhage are also common[15,17]. In the present study one of the patients was a primigravida and had a maipresentation. There was no known teratogenic exposure and no predisposing factor could be identified. Pregnancy proceded to term in both and although grave dystocia was encountered, third stage complications were not observed in any.
Fetal ascites demands early prenatal recognition. The condition has to be distinguished from other causes of fetal abdominal enlargement such as a distended bladder,[27,28], poly-cystic kidneys[29], fetal, ovarian cysts[30], and in rare instances from liver tumours, massive aortic aneurysms, inclusions like fetus in fetu and tumours from almost any where in the abdomen[6,31,32].
The diagnosis depends on a high degree of clinical suspicion and is confirmed by ultrasonographic evaluation which is the most reliable diagnostic tool[2,15,17,24]. A thorough workup is indicated to determine the underlying etiology. An immunologic cause must be excluded by antibody screening. Non-immunologic fetal ascites requires sonographic search for congenital malformations, serologic

testing for congenital infections, fetal echocardiography and heart rate monitoring and when appropriate haemoglobin electrophtrophoresis[2-4,15,16,21]. More invasive techniques like amniocentesis, fetoscopy and cordocentesis may be indicated for karyotyping, metabolic tests on fetal blood, DNA analysis and gene probe studies[16]. In our cases, although ascites was diagnosed on ultrasound examination, the cause remained undetermined because the cases were not adequately worked up. In the first case consanguineous marriage, the history of having delivered a malformed fetus, an abortion and a non immune hydrops fetalis with massive ascites in the current pregnancy were absolute indications for a thorough search into the etiological factors but the patient was lost to followup and reported back in labour. The second case was similarly seen in labour and henceforth also remained uninvestigated.

Fetal ascites carries an ominous prognosis with mortality almost approaching 98-100%[16,17]. Much depends upon the etiology. Transient cases, particularly those associated with infections, cardiac failure and lymphatic obstruction resolve rapidly[1,2,4,14]. It is only when there is persistence or progression that the fetal outcome is poor[21].

Treatment of fetal ascites is limited to a few situations only such as in selected cases of urinary tract obstruction and arrhythmias[15,16,21]. Management in labour is important in order to avoid dystocias. The unusual width of the fetal abdomen impedes progress and labour comes to a half Prompt decompression is required and as the abdomen approaches normal dimensions delivery is readily accomplished[5,7,8,31,32]. Pritchard et al[32] have described that paracentesis may be difficult because of the position of the fetus, edema of its abdominal wall and displacement of the liyer, but this is rarely encountered. Our first case unfortunately faced this situation and combined vaginal and abdominal delivery was required.

Fetal ascites has a low recurrence rate which varies from negligible to 25% in cases with specific modes of inheritance[15]. Genetic counselling, early recognition, close fetal surveillance, sound assessment during labour and timely delivery are recommended to avoid the risk of dystocias and to improve perinatal outcome.

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